The results of this study suggest that, among patients with ischaemic heart disease, spontaneous progenitor cell mobilisation is related to various mechanisms. On one hand, the higher concentration of progenitor cells in patients with chronic coronary artery disease than in controls (tonic mobilisation) indicates that coronary atherosclerosis alone is a sufficient trigger for haematopoietic progenitor cell mobilisation even in the absence of acute ischaemic stimuli. Indeed, compensatory progenitor cell mobilisation, phenotypically expressed by the percentage of circulating progenitor cells, appears to depend on a tonic process related to long lasting chronically active stimuli such as cardiovascular risk factors (hypertension, diabetes mellitus, family history) or chronic inflammation (C reactive protein). Tonic mobilisation of progenitor cells may be extremely helpful for the long term turnover and replenishment of a limited number of damaged vascular cells due to the atherosclerotic insult. Interestingly, major variability in the peak percentage of CD34+ cells was noted among patients with coronary artery disease, ranging from 0.04–1%, suggesting that the interplay between individual factors and environmental triggers has a major role in determining the magnitude of CD34+ tonic mobilisation. On the other hand, in the setting of acute myocardial ischaemia, when a variety of potentially haematopoietic progenitor cell mobilising factors are produced and released because of ischaemic damage, the higher number of circulating progenitor cells (phasic mobilisation) is achieved mainly by a non-specific increase in white blood cell production rather than by a specific increase in progenitor cell percentage. Notably, in the setting of acute myocardial ischaemia no correlation was found between peak concentration of circulating CD34+ cells and markers of myonecrosis suggesting that progenitor cell mobilisation is not correlated with specific ischaemia related triggers; rather, it depends on the non-specific mobilisation of leucocytes.

Authors' affiliations
A Gaspardone, F Menghini, V Mazzuca, O Skossyreva, G Barbato, Divisione di Cardiologia, Ospedale S Eugenio, Rome, Italy
P de Fabritiis, Ematologia, University Tor Vergata, Rome, Italy

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Correspondence to: Dr Achille Gaspardone, U O C di Cardiologia, Ospedale S Eugenio, ASL Rm C, Piazzale dell'Umanesimo 10, 00144 Rome, Italy; a_gaspardone@yahoo.com

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Computed tomographic pulmonary angiography and prognostic significance in patients with acute pulmonary embolism

A Ghuysen, B Ghaye, V Willems, B Lambermont, P Gerard, R F Dondelinger, V D'Orio

Please visit the *Heart* website [www.heartjnl.com] for a link to the full text of this article.

Background: Patients with acute pulmonary embolism (APE) present with a broad spectrum of prognoses. Computed tomographic pulmonary angiography (CTPA) has progressively been established as a first line test in the APE diagnostic algorithm, but estimation of short term prognosis by this method remains to be explored.
Methods: Eighty two patients admitted with APE were divided into three groups according to their clinical presentation: pulmonary infarction (n = 21), prominent dyspnoea (n = 29), and circulatory failure (n = 32). CTPA studies included assessment of both pulmonary obstruction index and right heart overload. Haemodynamic evaluation was based on systolic aortic blood pressure, heart rate, and systolic pulmonary arterial pressure obtained non-invasively by echocardiography at the time of diagnosis of pulmonary embolism.
Results: The mortality rate was 0%, 13.8% and 25% in the three groups, respectively. Neither the pulmonary obstruction index nor the pulmonary artery pressure could predict patient outcome. In contrast, a significant correlation with mortality was found using the systolic blood pressure ($p<0.001$) and heart rate ($p<0.05$), as well as from imaging parameters including right to left ventricle minor axis ratio ($p<0.005$), proximal superior vena cava diameter ($p<0.001$), azygos vein diameter ($p<0.001$), and presence of contrast regurgitation into the inferior vena cava ($p = 0.001$). Analysis from logistic regression aimed at testing for mortality prediction revealed true reclassification of 89% using radiological variables.
Conclusion: These results suggest that CTPA quantification of right ventricular strain is an accurate predictor of in-hospital death related to pulmonary embolism.

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